=> d his

(FILE 'HOME' ENTERED AT 16:23:33 ON 28 MAR 2002)

FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 16:25:37 ON 28 MAR 2002

L1 3595 S (RECEPTOR(5A)ADVANCED(W)GLYCATION(W)ENDPRODUCT) OR RAGE
L2 24 S INHIBITOR(7A)L1
L3 70970 S (TISSUE(5A)GROWTH) OR NEOINTIMAL(3A)FORMATION OR RESTENOSIS
L4 242 S (INHIBIT? OR SUPPRESS? OR DEMINISH OR DIMINISH)(7A)L1
L5 6 S L3 AND L4
L6 3 DUP REM L5 (3 DUPLICATES REMOVED)

=> d au ti so ab 1-3 16

L6 ANSWER 1 OF 3 MEDLINE DUPLICATE 1
AU Degryse B; Bonaldi T; Scaffidi P; Muller S; Resnati M; Sanvito F; Arrigoni G; Bianchi M E
TI The high mobility group (HMG) boxes of the nuclear protein HMG1 induce chemotaxis and cytoskeleton reorganization in rat smooth muscle cells.
SO JOURNAL OF CELL BIOLOGY, (2001 Mar 19) 152 (6) 1197-206.
Journal code: HMV; 0375356. ISSN: 0021-9525.
AB HMG1 (high mobility group 1) is a ubiquitous and abundant chromatin component. However, HMG1 can be secreted by activated macrophages and monocytes, and can act as a mediator of inflammation and endotoxic lethality. Here we document a role of extracellular HMG1 in cell migration. HMG1 (and its individual DNA-binding domains) stimulated migration of rat smooth muscle cells in chemotaxis, chemokinesis, and wound healing assays. HMG1 induced rapid and transient changes of cell shape, and actin cytoskeleton reorganization leading to an elongated polarized morphology typical of motile cells. These effects were **inhibited** by antibodies directed against the **receptor** of **advanced glycation endproducts**, indicating that the receptor of advanced glycation endproducts is the receptor mediating the HMG1-dependent migratory responses. Pertussis toxin and the mitogen-activated protein kinase kinase inhibitor PD98059 also blocked HMG1-induced rat smooth muscle cell migration, suggesting that a G(i/o) protein and mitogen-activated protein kinases are required for the HMG1 signaling pathway. We also show that HMG1 can be released by damage or necrosis of a variety of cell types, including endothelial cells. Thus, HMG1 has all the hallmarks of a molecule that can promote atherosclerosis and **restenosis** after vascular damage.

L6 ANSWER 2 OF 3 SCISEARCH COPYRIGHT 2002 ISI (R)
AU Zhou Z M (Reprint); Marso S P; Schmidt A M; Stern D M; Qu W; Forudi F; Wang K; Lincoff A M; Topol E J
TI Blockade of receptor for advanced glycation end-products (**RAGE**) **suppresses neointimal formation** in diabetic rat carotid artery injury model
SO CIRCULATION, (31 OCT 2000) Vol. 102, No. 18, Supp. [S], pp. 246-246. MA 1202.
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3621 USA.
ISSN: 0009-7322.

L6 ANSWER 3 OF 3 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AU Zhou, Zhong Min (1); Marso, Steven P.; Schmidt, Ann Marie; Stern, David M.; Qu, Wu; Forudi, Farhad; Wang, Kai; Lincoff, A. Michael; Topol, Eric J.

TI Blockade of receptor for advanced glycation end-products (**RAGE**) **suppresses neointimal formation** in diabetic rat carotid artery injury model.
SO Circulation, (October 31, 2000) Vol. 102, No. 18 Supplement, pp. II.246. print.
Meeting Info.: Abstracts from Scientific Sessions 2000 New Orleans, Louisiana, USA November 12-15, 2000
ISSN: 0009-7322.

=> s l1 and l3
L7 14 L1 AND L3

=> dup rem l7
PROCESSING COMPLETED FOR L7
L8 8 DUP REM L7 (6 DUPLICATES REMOVED)

=> d his

(FILE 'HOME' ENTERED AT 16:23:33 ON 28 MAR 2002)

FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 16:25:37 ON 28 MAR 2002
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L4 242 S (INHIBIT? OR SUPPRESS? OR DEMINISH OR DIMINISH)(7A)L1
L5 6 S L3 AND L4
L6 3 DUP REM L5 (3 DUPLICATES REMOVED)
L7 14 S L1 AND L3
L8 8 DUP REM L7 (6 DUPLICATES REMOVED)

=> d au ti so ab 1-8 l8

L8 ANSWER 1 OF 8 MEDLINE DUPLICATE 1
AU Twigg S M; Chen M M; Joly A H; Chakrapani S D; Tsubaki J; Kim H S; Oh Y; Rosenfeld R G
TI Advanced glycosylation end products up-regulate connective **tissue** **growth** factor (insulin-like **growth** factor-binding protein-related protein 2) in human fibroblasts: a potential mechanism for expansion of extracellular matrix in diabetes mellitus.
SO ENDOCRINOLOGY, (2001 May) 142 (5) 1760-9.
Journal code: EGZ; 0375040. ISSN: 0013-7227.
AB Expansion of extracellular matrix with fibrosis occurs in many tissues as part of the end-organ complications in diabetes, and advanced glycosylation end products (AGE) are implicated as one causative factor in diabetic **tissue** fibrosis. Connective **tissue** **growth** factor (CTGF), also known as insulin-like growth factor-binding protein-related protein-2 (IGFBP-rP2), is a potent inducer of extracellular matrix synthesis and angiogenesis and is increased in tissues from rodent models of diabetes. The aim of this study was to determine whether CTGF is up-regulated by AGE in vitro and to explore the cellular mechanisms involved. AGE treatment of primary cultures of nonfetal human dermal fibroblasts in confluent monolayer increased CTGF steady state messenger RNA (mRNA) levels in a time- and dose-dependent manner. In contrast, mRNAs for other IGFBP superfamily members, IGFBP-rP1 (mac 25) and IGFBP-3, were not up-regulated by AGE. The effect of the AGE BSA reagent on CTGF mRNA was due to nonenzymatic glycosylation of BSA and,

using neutralizing antisera to AGE and to the receptor for AGE, termed **RAGE**, was seen to be due to late products of nonenzymatic glycosylation and was partly mediated by **RAGE**. Reactive oxygen species as well as endogenous transforming growth factor-beta1 could not explain the AGE effect on CTGF mRNA. AGE also increased CTGF protein in the conditioned medium and cell-associated CTGF. Thus, AGE up-regulates the profibrotic and proangiogenic protein CTGF (IGFBP-rP2), a finding that may have significance in the development of diabetic complications.

L8 ANSWER 2 OF 8 MEDLINE DUPLICATE 2
AU Degryse B; Bonaldi T; Scaffidi P; Muller S; Resnati M; Sanvito F; Arrigoni G; Bianchi M E
TI The high mobility group (HMG) boxes of the nuclear protein HMG1 induce chemotaxis and cytoskeleton reorganization in rat smooth muscle cells.
SO JOURNAL OF CELL BIOLOGY, (2001 Mar 19) 152 (6) 1197-206.
Journal code: HMV; 0375356. ISSN: 0021-9525.
AB HMG1 (high mobility group 1) is a ubiquitous and abundant chromatin component. However, HMG1 can be secreted by activated macrophages and monocytes, and can act as a mediator of inflammation and endotoxic lethality. Here we document a role of extracellular HMG1 in cell migration. HMG1 (and its individual DNA-binding domains) stimulated migration of rat smooth muscle cells in chemotaxis, chemokinesis, and wound healing assays. HMG1 induced rapid and transient changes of cell shape, and actin cytoskeleton reorganization leading to an elongated polarized morphology typical of motile cells. These effects were inhibited by antibodies directed against the **receptor** of **advanced glycation endproducts**, indicating that the **receptor** of **advanced glycation endproducts** is the receptor mediating the HMG1-dependent migratory responses. Pertussis toxin and the mitogen-activated protein kinase kinase inhibitor PD98059 also blocked HMG1-induced rat smooth muscle cell migration, suggesting that a G(i/o) protein and mitogen-activated protein kinases are required for the HMG1 signaling pathway. We also show that HMG1 can be released by damage or necrosis of a variety of cell types, including endothelial cells. Thus, HMG1 has all the hallmarks of a molecule that can promote atherosclerosis and **restenosis** after vascular damage.

L8 ANSWER 3 OF 8 SCISEARCH COPYRIGHT 2002 ISI (R)
AU Fehrenbach H (Reprint); Weiskirchen R; Kasper M; Gressner A M
TI Up-regulated expression of the receptor for advanced glycation end products in cultured rat hepatic stellate cells during transdifferentiation to myofibroblasts
SO HEPATOLOGY, (NOV 2001) Vol. 34, No. 5, pp. 943-952.
Publisher: W B SAUNDERS CO, INDEPENDENCE SQUARE WEST CURTIS CENTER, STE 300, PHILADELPHIA, PA 19106-3399 USA.
ISSN: 0270-9139.
AB Receptor for advanced glycation end products (**RAGE**) is a member of the immunoglobulin superfamily of cell-surface molecules. Blockade of **RAGE** has been reported to considerably improve liver function and accelerate regeneration after hepatectomy. The aim of this study was to investigate the cell type-specific expression of **RAGE**, and to examine whether transdifferentiation of hepatic stellate cells (HSC) into myofibroblasts (MFB) is associated with changes in **RAGE** expression. Northern blot analysis revealed that **RAGE** mRNA was exclusively expressed by HSC isolated from rat liver, while no transcripts

were seen in hepatocytes, Kupffer cells, or sinusoidal endothelial cells. Expression of **RAGE** mRNA was up-regulated during transdifferentiation of HSC into MFB. Concomitantly, expression of **RAGE** protein was increased as confirmed by Western blotting and immunohistochemistry. As assessed by radioactive labeling, transforming growth factor beta (1) (TGF-beta (1)) induced a time-dependent 2- to 15-fold increase in the de novo synthesis of **RAGE** protein, which was completely abolished using PD098059, a specific inhibitor of the mitogen-activated protein kinase (MAPK) kinase. As shown by double-immunofluorescence staining, **RAGE** colocalized with a-smooth muscle actin, and immunoelectron microscopy demonstrated the most prominent labeling for **RAGE** at filopodial membranes of MFB. In conclusion, this study demonstrates that expression of **RAGE** is restricted to rat HSC, and that expression is up-regulated during activation of HSC and transition to MFB. The preferential immunogold labeling of **RAGE** to focal membrane areas of filopodia of MFB is suggestive of a role of **RAGE** in the spreading and migration of activated HSC/MFB, major players in liver fibrogenesis.

L8 ANSWER 4 OF 8 SCISEARCH COPYRIGHT 2002 ISI (R)
AU Sakaguchi T (Reprint); Sousa M; Yan S D; Yan S F; Duda S; Arnold B; Nawroth P P; Schmidt A M; Stern D M; Naka Y
TI **Restenosis**: Central role of **RAGE**-dependent neointimal expansion
SO CIRCULATION, (23 OCT 2001) Vol. 104, No. 17, Supp. [S], pp. 522-523. MA 2471.
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3621 USA.
ISSN: 0009-7322.

L8 ANSWER 5 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AU Kornberg, Abraham (1); Buchs, Andreas; Zahavi, Miriam; Rapoport, Micha
TI Increased tissue factor in monocytes form patients with diabetes mellitus.
SO Blood, (November 16, 2001) Vol. 98, No. 11 Part 2, pp. 28b.
http://www.bloodjournal.org/. print.
Meeting Info.: 43rd Annual Meeting of the American Society of Hematology, Part 2 Orlando, Florida, USA December 07-11, 2001
ISSN: 0006-4971.
AB Vascular complications such as arteriosclerosis develop frequently in patients with diabetes mellitus (DM). Advanced glycosylation end products (AGEs) play an important role in the development of the complications. The lesions contain in addition to lipids also fibrin, thrombin and platelets, indicating that the coagulation system is activated. The lesions contain also monocytes-macrophages that accumulate at an early stage of atheroma formation and fibroblasts and connective-tissue. Monocytes possess specific receptors for AGEs (**RAGE**). Cell-bound tissue factor (TF) is the main initiator of blood coagulation. TF also induce the expression of connective **tissue** and fibroblast **growth** factors. Monocytes, but not lymphocytes, can generate a potent TF upon stimulation with various substances. Monocyte TF is increased in diseases with high incidence of thrombosis. Based on this data the goal of the study was to investigate the capacity of monocytes from patients with DM with vascular complications (angina pectoris, myocardial infarction, stroke, peripheral vascular disease) and without to generate TF. Mononuclear cells (MNC) were isolated on Ficoll-hypaque gradient centrifugation and incubated with and without AGE-albumin for 18 hours.

Monocyte TF activity was assayed by a modified PT and TF antigen by American Diagnostica ELISA. For TF mRNA and **RAGE** mRNA determination, RNA was extracted, reversed transcribed into DNA and the genes were amplified and compared to the housekeeping gene GAPDH. The results show that TF and **RAGE** are increased significantly in cells from patients with DM and vascular complications. After stimulation with AGE-albumin TF increased significantly in MNC from patients without vascular complications and even more in patients with complications (2.1-3.2 fold; $p<0.07$). The results of the study suggest that monocyte TF play an important role in the pathogenesis of vascular complication in patients with DM and that AGEs may exert their effect via the induction of monocyte TF.

L8 ANSWER 6 OF 8 SCISEARCH COPYRIGHT 2002 ISI (R)
AU Zhou Z M (Reprint); Marso S P; Schmidt A M; Stern D M; Qu W; Forudi F; Wang K; Lincoff A M; Topol E J
TI Blockade of receptor for advanced glycation end-products (**RAGE**) suppresses **neointimal formation** in diabetic rat carotid artery injury model
SO CIRCULATION, (31 OCT 2000) Vol. 102, No. 18, Supp. [S], pp. 246-246. MA 1202.
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3621 USA.
ISSN: 0009-7322.

L8 ANSWER 7 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AU Zhou, Zhong Min (1); Marso, Steven P.; Schmidt, Ann Marie; Stern, David M.; Qu, Wu; Forudi, Farhad; Wang, Kai; Lincoff, A. Michael; Topol, Eric J.
TI Blockade of receptor for advanced glycation end-products (**RAGE**) suppresses **neointimal formation** in diabetic rat carotid artery injury model.
SO Circulation, (October 31, 2000) Vol. 102, No. 18 Supplement, pp. II.246. print.
Meeting Info.: Abstracts from Scientific Sessions 2000 New Orleans, Louisiana, USA November 12-15, 2000
ISSN: 0009-7322.

L8 ANSWER 8 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AU Vojacek, Jan (1); Simek, Stanislav; Aschermann, Michael; Krupicka, Petr
TI **Restenosis** after coronary angioplasty.
SO Cor et Vasa, (1995) Vol. 37, No. 6, pp. 342-347.
ISSN: 0010-8650.
AB The aim of the study was to analyze the causes of **restenosis** following coronary angioplasty in a group of patients hospitalized at the Department of Medicine II of the Prague-based Charles University School of Medicine 1, and to evaluate the outcome in patients undergoing repeat coronary arteriography for suspicion of coronary **restenosis**. In the period between 16 March 1989 and 17 November 1994, a total of 601 percutaneous transluminar coronary angioplasty procedures were performed in 489 patients (369 men, 120 women, age **rage** 31-85 years, mean age 54.0 +- 9.6 years). Over the same period of time. 116 follow-up coronary arteriography procedures were carried out in patients who had had a successful coronary angioplasty. While no **restenosis** was found in 50 cases, a **restenosis** was present in 66 cases. Of this number, repeat coronary angioplasty was performed in 55 patients, surgical

myocardial revascularization was attempted in seven, and four patients continued to receive conservative therapy. The success rate of coronary angioplasty in patients developing **restenosis** was 94.5% which is not only higher than in the basic group (with the overall success rate being 84.1% over the same follow-up period, regardless of the type of coronary stenosis) but, also, higher than in the basic group of patients with Type A coronary stenosis. Six patients had a third coronary angioplasty procedure in the same localization with success in four; a fourth angioplasty was successful in yet another patient. **Restenosis** was more frequent in stenoses with a suboptimal outcome immediately after angioplasty than in those achieving optimal dilatation (57.7% vs 47.4% of patients with follow-up coronary arteriography); the difference, however, was not statistically significant. Although experimental results may seem to suggest a number of promising procedures are currently available, repeat coronary angioplasty remains to be the most reliable method for treating restenoses in clinical practice. Coronary stent implantation reduces the risk of **restenosis** in some patients.

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=> s l1 and (diabetes or diabetic)
L9          492 L1 AND (DIABETES OR DIABETIC)

=> s l4 and (diabetes or diabetic)
L10          91 L4 AND (DIABETES OR DIABETIC)

=> dup rem l10
PROCESSING COMPLETED FOR L10
L11          40 DUP REM L10 (51 DUPLICATES REMOVED)

=> d his

     (FILE 'HOME' ENTERED AT 16:23:33 ON 28 MAR 2002)

     FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 16:25:37 ON 28 MAR
     2002
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L2           24 S INHIBITOR(7A)L1
L3        70970 S (TISSUE(5A)GROWTH) OR NEOINTIMAL(3A)FORMATION OR RESTENOSIS
L4          242 S (INHIBIT? OR SUPPRESS? OR DEMINISH OR DIMINISH)(7A)L1
L5            6 S L3 AND L4
L6            3 DUP REM L5 (3 DUPLICATES REMOVED)
L7           14 S L1 AND L3
L8            8 DUP REM L7 (6 DUPLICATES REMOVED)
L9          492 S L1 AND (DIABETES OR DIABETIC)
L10          91 S L4 AND (DIABETES OR DIABETIC)
L11          40 DUP REM L10 (51 DUPLICATES REMOVED)

=> d au ti so 1-40 l11

L11  ANSWER 1 OF 40     MEDLINE
AU   Collison Kate S; Parhar Ranjit S; Saleh Soad S; Meyer Brian F; Kwaasi
     Aaron A; Hammami Muhammad M; Schmidt Ann Marie; Stern David M; Al-Mohanna
     Futwan A
TI   RAGE-mediated neutrophil dysfunction is evoked by advanced glycation end
     products (AGEs).
SO   JOURNAL OF LEUKOCYTE BIOLOGY, (2002 Mar) 71 (3) 433-44.
     Journal code: 8405628. ISSN: 0741-5400.

L11  ANSWER 2 OF 40  SCISEARCH  COPYRIGHT 2002 ISI (R)
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AU Boulanger E; Wautier M P (Reprint); Wautier J L; Boval B; Panis Y; Wernert
N; Danze P M; Dequiedt P
TI AGEs bind to mesothelial cells via RAGE and stimulate VCAM-1 expression
SO KIDNEY INTERNATIONAL, (JAN 2002) Vol. 61, No. 1, pp. 148-156.
Publisher: BLACKWELL SCIENCE INC, 350 MAIN ST, MALDEN, MA 02148 USA.
ISSN: 0085-2538.

L11 ANSWER 3 OF 40 CAPLUS COPYRIGHT 2002 ACS
AU Lim, Hyun Jin; Song, Jaesook; Ha, Hunjoo; Lee, Hi Bahl
TI N.epsilon.-(carboxymethy)lysine-induced mesangial cell activation
SO Taehan Sinjang Hakhoechi (2002), 21(1), 20-28
CODEN: TSHACY; ISSN: 1225-0015

L11 ANSWER 4 OF 40 CAPLUS COPYRIGHT 2002 ACS
IN Shahbaz, Manouchehr
TI Methods to identify compounds that modulate RAGE
SO PCT Int. Appl., 49 pp.
CODEN: PIXXD2

L11 ANSWER 5 OF 40 MEDLINE DUPLICATE 1
AU Yeh C H; Sturgis L; Haidacher J; Zhang X N; Sherwood S J; Bjercke R J; Juhasz O; Crow M T; Tilton R G; Denner L
TI Requirement for p38 and p44/p42 mitogen-activated protein kinases in RAGE-mediated nuclear factor-kappaB transcriptional activation and cytokine secretion.
SO DIABETES, (2001 Jun) 50 (6) 1495-504.
Journal code: E8X; 0372763. ISSN: 0012-1797.

L11 ANSWER 6 OF 40 MEDLINE DUPLICATE 2
AU Schmidt A M; Stern D M
TI Receptor for age (RAGE) is a gene within the major histocompatibility class III region: implications for host response mechanisms in homeostasis
and chronic disease.
SO FRONTIERS IN BIOSCIENCE, (2001 Oct 1) 6 D1151-60. Ref: 48
Journal code: 9702166. ISSN: 1093-4715.

L11 ANSWER 7 OF 40 MEDLINE DUPLICATE 3
AU Goova M T; Li J; Kislinger T; Qu W; Lu Y; Bucciarelli L G; Nowygrod S; Wolf B M; Caliste X; Yan S F; Stern D M; Schmidt A M
TI Blockade of receptor for advanced glycation end-products restores effective wound healing in **diabetic** mice.
SO AMERICAN JOURNAL OF PATHOLOGY, (2001 Aug) 159 (2) 513-25.
Journal code: 3RS; 0370502. ISSN: 0002-9440.

L11 ANSWER 8 OF 40 MEDLINE DUPLICATE 4
AU Wang R; Kudo M; Yokoyama M; Asano G
TI Roles of advanced glycation endproducts (AGE) and receptor for AGE on vascular smooth muscle cell growth.
SO JOURNAL OF NIPPON MEDICAL SCHOOL, (2001 Dec) 68 (6) 472-81.
Journal code: 100935589. ISSN: 1345-4676.

L11 ANSWER 9 OF 40 SCISEARCH COPYRIGHT 2002 ISI (R)
AU Wendt T M (Reprint); Tanji N; Kislinger T; Bucciarelli L G; Qu W; Lu Y; Lalla E; Moser B; Markowitz G; D'Agati V; Stern D M; Schmidt A M
TI Blockade of receptor for age (**RAGE**) **suppresses** albuminuria and glomerulosclerosis in murine **diabetic** kidney: Implications for podocyte activation in the pathogenesis of **diabetic** nephropathy

SO CIRCULATION, (23 OCT 2001) Vol. 104, No. 17, Supp. [S], pp. 237-237. MA 1142.
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3621 USA.
ISSN: 0009-7322.

L11 ANSWER 10 OF 40 MEDLINE DUPLICATE 5
AU Huang J S; Guh J Y; Chen H C; Hung W C; Lai Y H; Chuang L Y
TI Role of receptor for advanced glycation end-product (RAGE) and the JAK/STAT-signaling pathway in AGE-induced collagen production in NRK-49F cells.
SO JOURNAL OF CELLULAR BIOCHEMISTRY, (2001) 81 (1) 102-13.
Journal code: HNF; 8205768. ISSN: 0730-2312.

L11 ANSWER 11 OF 40 CAPLUS COPYRIGHT 2002 ACS
IN Schmidt, Ann Marie; Stern, David
TI Methods for determining whether a compound is capable of **inhibiting** the interaction of a peptide with **RAGE**
SO PCT Int. Appl., 66 pp.
CODEN: PIXXD2

L11 ANSWER 12 OF 40 MEDLINE DUPLICATE 6
AU Lalla E; Lamster I B; Feit M; Huang L; Spessot A; Qu W; Kislinger T; Lu Y;
Stern D M; Schmidt A M
TI Blockade of **RAGE suppresses** periodontitis-associated bone loss in **diabetic** mice.
SO JOURNAL OF CLINICAL INVESTIGATION, (2000 Apr) 105 (8) 1117-24.
Journal code: HS7; 7802877. ISSN: 0021-9738.

L11 ANSWER 13 OF 40 SCISEARCH COPYRIGHT 2002 ISI (R)
AU Yan S D; Zhu H J; Zhu A P; Golabek A; Du H; Roher A; Yu J; Soto C; Schmidt
A M; Stern D; Kindy M (Reprint)
TI Receptor-dependent cell stress and amyloid accumulation in systemic amyloidosis
SO NATURE MEDICINE, (JUN 2000) Vol. 6, No. 6, pp. 643-651.
Publisher: NATURE AMERICA INC, 345 PARK AVE SOUTH, NEW YORK, NY 10010-1707.
ISSN: 1078-8956.

L11 ANSWER 14 OF 40 MEDLINE DUPLICATE 7
AU Taguchi A; Blood D C; del Toro G; Canet A; Lee D C; Qu W; Tanji N; Lu Y; Lalla E; Fu C; Hofmann M A; Kislinger T; Ingram M; Lu A; Tanaka H; Hori O;
Ogawa S; Stern D M; Schmidt A M
TI Blockade of **RAGE**-amphoterin signalling **suppresses** tumour growth and metastases.
SO NATURE, (2000 May 18) 405 (6784) 354-60.
Journal code: NSC; 0410462. ISSN: 0028-0836.

L11 ANSWER 15 OF 40 SCISEARCH COPYRIGHT 2002 ISI (R)
AU Zhou Z M (Reprint); Marso S P; Schmidt A M; Stern D M; Qu W; Forudi F; Wang K; Lincoff A M; Topol E J
TI Blockade of receptor for advanced glycation end-products (**RAGE**) **suppresses** neointimal formation in **diabetic** rat carotid artery injury model
SO CIRCULATION, (31 OCT 2000) Vol. 102, No. 18, Supp. [S], pp. 246-246. MA 1202.
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA

19106-3621 USA.
ISSN: 0009-7322.

L11 ANSWER 16 OF 40 SCISEARCH COPYRIGHT 2002 ISI (R)
AU Bucciarelli L G (Reprint); Qu W; Lu Y; Wendt T M; Kislinger T R; Goova M T; Ferran L A; Stern D M; Schmidt A M
TI Blockade of receptor for AGE (**RAGE**) **suppresses** progression of established atherosclerotic lesions in APO E null mice with type 1 **diabetes**.
SO CIRCULATION, (31 OCT 2000) Vol. 102, No. 18, Supp. [S], pp. 232-232. MA 1128.
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3621 USA.
ISSN: 0009-7322.

L11 ANSWER 17 OF 40 MEDLINE DUPLICATE 8
AU Bonnardel-Phu E; Wautier J L; Vicaut E
TI [Advanced glycation end products are involved in microvascular permeability changes observed in microcirculation of **diabetic** rats in vivo].
Les produits avances de la glycation sont impliques dans les changements de la permeabilite microvasculaire observes chez le rat diabetique in vivo.
SO JOURNAL DES MALADIES VASCULAIRES, (2000 Apr) 25 (2) 122-7.
Journal code: IYN; 7707965. ISSN: 0398-0499.

L11 ANSWER 18 OF 40 SCISEARCH COPYRIGHT 2002 ISI (R)
AU Bucciarelli L G (Reprint); Qu W; Wendt T M; Goova M T; Bakr S; Hwang Y Y C; Stern D M; Schmidt A M; Ramasamy R
TI Blockade of receptor for AGE (**RAGE**) **suppresses** levels of cardiac endothelial- and inducible nitric oxide synthase in **diabetic** mice
SO CIRCULATION, (31 OCT 2000) Vol. 102, No. 18, Supp. [S], pp. 117-118. MA 563.
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3621 USA.
ISSN: 0009-7322.

L11 ANSWER 19 OF 40 MEDLINE DUPLICATE 9
AU Schmidt A M; Yan S D; Yan S F; Stern D M
TI The biology of the receptor for advanced glycation end products and its ligands.
SO BIOCHIMICA ET BIOPHYSICA ACTA, (2000 Dec 20) 1498 (2-3) 99-111. Ref: 42
Journal code: A0W. ISSN: 0006-3002.

L11 ANSWER 20 OF 40 SCISEARCH COPYRIGHT 2002 ISI (R)
AU Kislinger T R (Reprint); Tanji N; Qu W; Goova M T; Wendt T M; Lu Y; Bucciarelli L G; Hofmann M A; Ferran L A; Pischetsrieder M; Stern D M; Schmidt A M
TI Blockade of receptor for AGE (**RAGE**) **suppresses** vascular inflammation and hypercoagulability in apo E null mice with type 1 **diabetes**.
SO CIRCULATION, (31 OCT 2000) Vol. 102, No. 18, Supp. [S], pp. 41-41. MA 187.
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3621 USA.
ISSN: 0009-7322.

L11 ANSWER 21 OF 40 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

AU Zhou, Zhong Min (1); Marso, Steven P.; Schmidt, Ann Marie; Stern, David M.; Qu, Wu; Forudi, Farhad; Wang, Kai; Lincoff, A. Michael; Topol, Eric J.
TI Blockade of receptor for advanced glycation end-products (**RAGE**) **suppresses** neointimal formation in **diabetic** rat carotid artery injury model.
SO Circulation, (October 31, 2000) Vol. 102, No. 18 Supplement, pp. II.246. print.
Meeting Info.: Abstracts from Scientific Sessions 2000 New Orleans, Louisiana, USA November 12-15, 2000
ISSN: 0009-7322.

L11 ANSWER 22 OF 40 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AU Kislinger, Thomas R. (1); Tanji, Nozomu (1); Qu, Wu (1); Goova, Mouza T. (1); Wendt, Thoralf M. (1); Lu, Yan (1); Bucciarelli, Loredana G. (1); Hofmann, Marion A. (1); Ferran, Luis A. (1); Pischetsrieder, Monika; Stern, David M.; Schmidt, Ann Marie
TI Blockade of receptor for AGE (**RAGE**) **suppresses** vascular inflammation and hypercoagulability in apo E null mice with type 1 **diabetes**.
SO Circulation, (October 31, 2000) Vol. 102, No. 18 Supplement, pp. II.41. print.
Meeting Info.: Abstracts from Scientific Sessions 2000 New Orleans, Louisiana, USA November 12-15, 2000
ISSN: 0009-7322.

L11 ANSWER 23 OF 40 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AU Bucciarelli, Loredana G. (1); Qu, Wu (1); Wendt, Thoralf M. (1); Goova, Mouza T. (1); Bakr, Soliman (1); Hwang, Yuying C. (1); Stern, David M. (1); Schmidt, Ann Marie (1); Ramasamy, Ravichandran (1)
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SO Circulation, (October 31, 2000) Vol. 102, No. 18 Supplement, pp. II.117-II.118. print.
Meeting Info.: Abstracts from Scientific Sessions 2000 New Orleans, Louisiana, USA November 12-15, 2000
ISSN: 0009-7322.

L11 ANSWER 24 OF 40 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AU Bucciarelli, Loredana G. (1); Qu, Wu (1); Lu, Yan (1); Wendt, Thoralf M. (1); Kislinger, Thomas R. (1); Goova, Mouza T. (1); Ferran, Luis A. (1); Stern, David M. (1); Schmidt, Ann Marie (1)
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SO Circulation, (October 31, 2000) Vol. 102, No. 18 Supplement, pp. II.232. print.
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SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1999 Oct 29) 274 (44) 31740-9.
Journal code: HIV; 2985121R. ISSN: 0021-9258.

L11 ANSWER 26 OF 40 MEDLINE DUPLICATE 11
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SO JOURNAL OF PHARMACOLOGY AND EXPERIMENTAL THERAPEUTICS, (1999 Sep) 290 (3) 1458-66.
Journal code: JP3; 0376362. ISSN: 0022-3565.

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SO CIRCULATION RESEARCH, (1999 Mar 19) 84 (5) 489-97. Ref: 89
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L11 ANSWER 28 OF 40 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE
13
AU Salahudeen, A. K. (1); Huang, H. (1); Stern, D.; Schmidt, A. M.
TI Administration of soluble **receptor** for **advanced glycation endproducts** (sRAGE) in DB-DB mice **suppresses** abnormalities in the early and late stages of **diabetic** nephropathy.
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ISSN: 0892-6638.

L11 ANSWER 29 OF 40 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE
14
AU Salahudeen, A. K. (1); Huang, H. (1); Stern, D.; Schmidt, A. M.
TI Administration of soluble **receptor** for **advanced glycation endproducts** (sRAGE) in DB-DB mice **suppresses** abnormalities in the early and late stages of **diabetic** nephropathy.
SO Journal of Investigative Medicine, (April, 1999) Vol. 47, No. 4, pp. 207A.
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. ISSN: 1081-5589.

L11 ANSWER 30 OF 40 CAPLUS COPYRIGHT 2002 ACS
IN Stern, David M.; Schmidt, Ann Marie
TI Method for treating symptoms of **diabetes** with agents preventing binding of advanced glycation endproducts to receptors
SO PCT Int. Appl., 33 pp.
CODEN: PIXXD2

L11 ANSWER 31 OF 40 SCISEARCH COPYRIGHT 2002 ISI (R)
AU Li J F; Qu X Q; Schmidt A M (Reprint)
TI Sp1-binding elements in the promoter of RAGE are essential for amphoterin-mediated gene expression in cultured neuroblastoma cells

SO JOURNAL OF BIOLOGICAL CHEMISTRY, (20 NOV 1998) Vol. 273, No. 47, pp. 30870-30878.
Publisher: AMER SOC BIOCHEMISTRY MOLECULAR BIOLOGY INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814.
ISSN: 0021-9258.

L11 ANSWER 32 OF 40 MEDLINE DUPLICATE 15
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TI **Suppression** of accelerated **diabetic** atherosclerosis by the soluble **receptor** for **advanced glycation endproducts**.
SO NATURE MEDICINE, (1998 Sep) 4 (9) 1025-31.
Journal code: CG5; 9502015. ISSN: 1078-8956.

L11 ANSWER 33 OF 40 CAPLUS COPYRIGHT 2002 ACS
IN Morser, Michael John; Nagashima, Mariko; Hollander, Doris Anne
TI Antibodies against the advanced glycosylation end-product receptor and uses thereof
SO PCT Int. Appl., 89 pp.
CODEN: PIXXD2

L11 ANSWER 34 OF 40 CAPLUS COPYRIGHT 2002 ACS
IN Morser, Michael John; Nagashima, Mariko
TI Advanced glycosylation end-product receptor peptides and their uses for increasing vascular permeability in disease conditions
SO PCT Int. Appl., 91 pp.
CODEN: PIXXD2

L11 ANSWER 35 OF 40 SCISEARCH COPYRIGHT 2002 ISI (R)
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TI A murine model of accelerated **diabetic** atherosclerosis: **Suppression** by soluble **receptor** for **advanced glycation endproducts**
SO CIRCULATION, (21 OCT 1997) Vol. 96, No. 8, Supp. [S], pp. 3079-3079.
Publisher: AMER HEART ASSOC, 7272 GREENVILLE AVENUE, DALLAS, TX 75231-4596.
ISSN: 0009-7322.

L11 ANSWER 36 OF 40 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 16
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TI Advanced glycation end product (AGE)-mediated induction of tissue factor in cultured endothelial cells is dependent on RAGE.
SO Circulation, (1997) Vol. 96, No. 7, pp. 2262-2271.
ISSN: 0009-7322.

L11 ANSWER 37 OF 40 MEDLINE DUPLICATE 17
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TI Receptor-mediated endothelial cell dysfunction in **diabetic** vasculopathy. Soluble receptor for advanced glycation end products blocks hyperpermeability in **diabetic** rats.
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Journal code: HS7; 7802877. ISSN: 0021-9738.

L11 ANSWER 38 OF 40 MEDLINE DUPLICATE 18
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SO NEPHROLOGY, DIALYSIS, TRANSPLANTATION, (1996) 11 Suppl 5 13-6. Ref: 26
Journal code: N7J; 8706402. ISSN: 0931-0509.

L11 ANSWER 39 OF 40 MEDLINE DUPLICATE 19
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SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1995 Oct 27) 270 (43) 25752-61.
Journal code: HIV; 2985121R. ISSN: 0021-9258.

L11 ANSWER 40 OF 40 MEDLINE DUPLICATE 20
AU Schmidt A M; Hori O; Chen J X; Li J F; Crandall J; Zhang J; Cao R; Yan S D; Brett J; Stern D
TI Advanced glycation endproducts interacting with their endothelial receptor
induce expression of vascular cell adhesion molecule-1 (VCAM-1) in cultured human endothelial cells and in mice. A potential mechanism for the accelerated vasculopathy of **diabetes**.
SO JOURNAL OF CLINICAL INVESTIGATION, (1995 Sep) 96 (3) 1395-403.
Journal code: HS7; 7802877. ISSN: 0021-9738.

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=> s l4 and (stroke or angina or thrombosis or infarction)
L12 2 L4 AND (STROKE OR ANGINA OR THROMBOSIS OR INFARCTION)

=> dup rem l12
PROCESSING COMPLETED FOR L12
L13 2 DUP REM L12 (0 DUPLICATES REMOVED)

=> d au ti so ab 1-2 l13

L13 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2002 ACS
IN Stern, David M.; Schmidt, Ann Marie; Yan, Shi Du; Zlokovic, Berislav
TI A method to increase cerebral blood flow in cerebral amyloid angiopathy by
administering to the subject an inhibitor of receptor for advanced glycosylation end product
SO PCT Int. Appl., 68 pp.
CODEN: PIXXD2
AB The present invention provides a method for decreasing cerebral vasoconstriction in a subject suffering from chronic or acute cerebral amyloid angiopathy which comprises administering to the subject an **inhibitor** of receptor for advanced glycation end product (**RAGE**) in an effective amt. to **inhibit** transcytosis of amyloid .beta. peptides across the blood-brain barrier in the subject, thereby decreasing cerebral vasoconstriction in the subject. The invention further provides for a method for ameliorating neurovascular stress in a subject which comprises administering to the subject an effective amt. of an **inhibitor** of receptor for advanced glycation end product (**RAGE**), so as to increase cerebral blood flow in the subject, thereby ameliorating neurovascular stress in the subject. The invention demonstrates that RAGE has import role in A.beta.-mediated uptake at blood-brain barrier (BBB) and its transport into the central nervous system, as well as a.beta.-mediated cellular perturbation. A method for blockading RAGE, with either sol. **RAGE** or anti-**RAGE** IgG which, thereby **suppresses** binding to and uptake of a.beta. in relation to the vessel wall and inhibits a.beta.-induced cell stress in the vasculature and in neurons, consequent to systemic infusion of a.beta..

L13 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2002 ACS
IN Stern, David M.; Schmidt, Ann Marie
TI Method for treating symptoms of diabetes with agents preventing binding of
advanced glycation endproducts to receptors
SO PCT Int. Appl., 33 pp.
CODEN: PIXXD2
AB A method is provided for treating symptoms of diabetes in a diabetic subject, e.g. abnormal wound healing, which comprises administering to the
subject a therapeutically effective amt. of an agent which **inhibits** binding of advanced glycation endproducts to any **receptor** for **advanced glycation endproducts** so as to treat chronic symptoms of diabetes in the subject. Improved wound healing in diabetic mice by treatment with the sol. receptor for advanced glycation endproducts is described.

=> d bib 1-2 l13

L13 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2002 ACS

AN 2002:142891 CAPLUS
DN 136:178004
TI A method to increase cerebral blood flow in cerebral amyloid angiopathy by
administering to the subject an inhibitor of receptor for advanced glycosylation end product
IN Stern, David M.; Schmidt, Ann Marie; Yan, Shi Du; Zlokovic, Berislav
PA The Trustees of Columbia University in the City of New York, USA
SO PCT Int. Appl., 68 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002014519	A1	20020221	WO 2001-US25416	20010814

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 2000-638648 A 20000814
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ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2002 ACS
AN 1998:351788 CAPLUS
DN 129:12747
TI Method for treating symptoms of diabetes with agents preventing binding of
advanced glycation endproducts to receptors
IN Stern, David M.; Schmidt, Ann Marie
PA Trustees of Columbia University in the City of New York, USA
SO PCT Int. Appl., 33 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9822138	A1	19980528	WO 1997-US21197	19971112
W: AU, CA, JP, MX				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9852639	A1	19980610	AU 1998-52639	19971112
EP 946196	A1	19991006	EP 1997-947592	19971112
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2001504493	T2	20010403	JP 1998-523860	19971112
PRAI US 1996-755235	A	19961122		
WO 1997-US21197	W	19971112		

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L11 ANSWER 33 OF 40 CAPLUS COPYRIGHT 2002 ACS
AN 1997:696868 CAPLUS
DN 128:2908
TI Antibodies against the advanced glycosylation end-product receptor and uses thereof
IN Morser, Michael John; Nagashima, Mariko; Hollander, Doris Anne
PA Schering Aktiengesellschaft Patente, Germany
SO PCT Int. Appl., 89 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9739125	A1	19971023	WO 1997-EP1834	19970411
	W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
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